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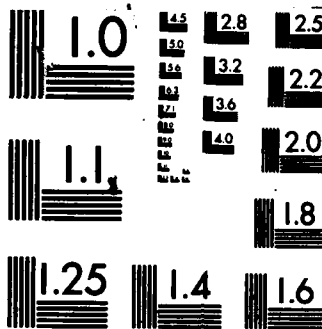UNCLASSIFIED   N00014-83-K-0019   F/G 6/19   NL

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20. ABSTRACT (Continue on reverse side if necessary and identify by block number)

(I) A theoretical method has been developed to predict space craft and space suit atmospheres. The method was used to analyze stage decompression procedures which have been tested for the Shuttle. (II) The effect of exercise during compression and bottom time has been studied with rats during an 18 minute air dive at 350 fsw. Seventeen resting rats and 27 exercising rats out of 2 groups of 108 rats developed decompression sickness. (III) A method for developing decompression schedules for helium/oxygen and nitrogen/oxygen saturation dives

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is described. Only one unknown constant is required for each saturation depth and inert gas, and this constant can be estimated from the results of previous saturation dives. Equations are presented for analyzing previous dives and for calculating decompression schedules, and examples of provisional schedules are given.

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DECOMPRESSION MECHANISMS AND
DECOMPRESSION SCHEDULE CALCULATIONS

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January 20, 1984

I. DECOMPRESSION IN SPACE

The theoretical methods developed under an earlier Navy contract and reported elsewhere (Vann 1982) were extended to decompression problems in space stations, space suits, and the space Shuttle. An abstract describing this work has been submitted for presentation at the 1984 Annual Meeting of the Undersea Medical Society, and a manuscript has been submitted by for publication to Av., Sp., and Environ. Med. (Vann and Torre-Bueno 1984). Copies of the abstract and manuscript are enclosed.

II. EXERCISE AND DECOMPRESSION

This study used rats to investigate the effect of exercise during bottom time on the incidence of decompression sickness. A standard test dive was selected from a series of no-decompression trials on air at 350 fsw. For each trial, a cage of 10 rats was placed in a 390 cuft. chamber. The rats were permitted to huddle together, and the following observations were made:

Bottom Time, min	No. DCS Cases	No. Rats	% DCS
14	1	10	10
15	1	10	10
16	7	29	24
18	8	20	40
20	16	31	52
21	6	11	55
22	8	10	80
25	8	10	80
30	8	10	80
45	7	10	70
60	9	10	90
120	10	10	100

From these trials, a bottom time of 18 minutes was selected as this gave a decompression sickness incidence of below 50%. Six rats were placed in separate compartments of a rotating cage in a 10 cuft. chamber. In the control dives, the rats remained at rest. In the experimental dives, the rotator turned at 24 rpm during decompression and bottom time. One hundred and eight (108) rats were tested under each condition. Fatal decompression sickness occurred for 17 resting rats (16%) and for 27 exercising rats (25%). Statistical analysis of these results is not yet complete.

III. A METHOD OF CALCULATING DECOMPRESSION SCHEDULES FOR NITROGEN/OXYGEN AND HELIUM/OXYGEN SATURATION DIVES

During the early 1970's, about 20 helium/oxygen man-dives were conducted at the F.G. Hall Lab to depths of 720 to 1000 fsw for bottom times of up to 4 hours. Decompression took place on a modified Buhlmann schedule with an inspired oxygen partial pressure (PIO2) of 0.8 ATM. Decompression sickness was rare, but pulmonary oxygen toxicity forced a reduction in PIO2 to 0.6 ATM. In subsequent dives at this lower PIO2 with the same schedule, there was an increase in the incidence of decompression sickness. To reduce this incidence, it was found necessary to use slower rates of ascent.

Similar observations with lower PIO2's were made in England at RNPL (Vorosmarti, Hanson, and Barnard 1978). Decompression schedules for saturation dives to 180 meters which were unsafe at a PIO2 of 0.22 ATM were found to be safe when the PIO2 was raised to 0.4 ATM.

References to the importance of the oxygen partial pressure can be found elsewhere in the literature. Experiments with gas pockets in animals showed that the rate of elimination of inert gas is proportional to the PIO2 (Van Liew et al. 1965). Decompression theory also predicted that the rate of ascent is proportional to the PIO2 (Workman 1969).

If it is assumed as experiment suggests and theory predicts that the ascent rate from a saturation dive is proportional to the PIO2, then

$$R = K*PIO2 \qquad (1)$$

where R is the rate of ascent in fsw per hour (fph), PIO2 is the inspired oxygen partial pressure in ATM, and K is a constant of proportionality in fph/ATM. From this relationship, it can be found that a decompression schedule for constant PIO2 is defined by

$$D(t) = Ds - K*PIO2*t \qquad (2)$$

where Ds is the saturation depth in fsw and t is time in hours. If the inspired oxygen fraction (FIO2) is constant,

$$D(t) = (Ds + 33)*exp(-K*FIO2*t/33) - 33 \qquad (3)$$

Saturation decompression schedules, therefore, are completely defined by the constant K.

While there is presently no method for predicting K, an estimated value, Ke, can be determined for saturation dives that have been conducted in the past. For example, when PIO2 is constant, Ke is given by

$$Ke = \frac{Ds - Ddcs}{PIO2*T}$$

where Ddcs is the depth at which symptoms of decompression sickness first occurred (or the surface if they did not occur) and T is the travel time in hours between the saturation depth Ds and Ddcs. When FIO2 is constant,

$$Ke = -\frac{33}{FIO2*T} * \ln(\frac{Ddcs - 33}{Ds - 33})$$

These values are only estimates of K as saturation decompression schedules are rarely completely linear or completely exponential.

Values of Ke are calculated for all well documented dives. Ke values are rejected as unsafe for dives resulting in decompression sickness. Ke values for dives not resulting in decompression sickness are accepted as safe only for schedules which have been used at least 30 times. The reliability of schedules with fewer trials is questionable because of statistical uncertainty.

Shortly before the Atlantis III dive at Duke, values of Ke were determined for 579 helium/oxygen decompressions, and it was found that decompression sickness had occurred only when Ke was greater than 10 fph/ATM (Vann and Dick 1981). For the Atlantis III dive to a depth of 2250 fsw, a K-value of 8 fph/ATM was used which resulted in a decompression schedule with an ascent rate of 4 fph at a PIO2 of 0.5 ATM. Two of 3 divers developed Type I decompression sickness with this schedule at depths of greater than 1700 fsw.

It was initially thought that these cases were caused by the 10% nitrogen in the breathing gas. With 5% nitrogen during the Atlantis IV decompression from 2132 fsw, however, decompression sickness occurred at 1412 fsw in 1 of 3 divers with a Ke of only 5.2 fph/ATM. These results indicated that nitrogen was probably not responsible for the decompression sickness and suggested that the safe value of K might decrease as the depth increased. A similar depth effect appears to exist for nitrogen/oxygen saturation decompression. This is discussed in two manuscripts recently submitted to Undersea Biomedical Research (Eckenhoff and Vann 1984; Barry, Vann, Youngblood, Peterson, and Bennett 1984). Copies of these man-

uscripts are enclosed.

The effect of depth on Ke is shown in Figs. 1 and 2 for both helium/oxygen and nitrogen/oxygen. Figure 1 shows the results of 1055 helium/oxygen man-decompressions during which there were 104 cases of decompression sickness. All the points above the solid line represent schedules with at least one case of decompression sickness. The points below the line represent schedules with at least 30 trials and no decompression sickness (58, 490, and 33 trials for these schedules). The solid line is an estimate of how K might vary with depth.

Figure 2 shows the results of 189 nitrogen/oxygen man-decompressions during which there were 38 cases of decompression sickness. As before, the solid line is an estimate of how K might vary with depth, and the points above this line represent schedules with at least one case of decompression sickness. There were no schedules without decompression sickness with at least 30 trials.

Figure 3 shows the helium/oxygen and nitrogen/oxygen data together. This figure can be used to illustrate an empirical method for developing saturation decompression schedules. Schedules are first calculated for both gases using equations (2) or (3) and the estimated K-lines shown in Fig. 3. If a schedule for a given depth causes decompression sickness, the K-value for that depth is adjusted downward, and a slower schedule is calculated. This procedure is repeated for each depth until the safe values of K are defined. The advantage of this method is that only one constant must be

changed for a given depth and inert gas to make a schedule more conservative. There is no guarantee, of course, that the resulting schedules will be the fastest or most efficient.

The estimated K-lines have been used to generate saturation decompression schedules for helium/oxygen dives from 2000 fsw (600 msw) and for nitrogen/oxygen dives from 200 fsw (60 msw). These schedules are shown in Tables 1 to 4. The PIO2 is held at 0.5 ATM from the maximum depth to 45 fsw (14 msw) after which the FIO2 is held at 0.21 until the surface. While the FIO2 is constant, the ascent rate is reduced as required by equation (1) to compensate for the falling oxygen partial pressure. For convenience, the ascent rate is reduced at 15 or 10 fsw intervals to the rate required by the lowest PIO2 in the interval. A rapid ascent is permitted for the first 10 fsw (3 msw) because of the oxygen window. For helium/oxygen diving, nitrogen is limited to not more than 5% deeper than 500 fsw (150 msw) and not more than 0.79 ATM shallower than 500 fsw.

Tables 1 to 4 were developed for a PIO2 of 0.5 ATM. At higher PIO2's, faster ascent rates should be possible. Above a partial pressure of 0.5 ATM, however, pulmonary oxygen toxicity develops, and its onset becomes more rapid as the PIO2 increases. Thus, the maximum permissible PIO2 decreases as the depth increases.

The material discussed above will be presented at the Third Annual Canadian Ocean Technology Congress in Toronto on 22-23 March 1934.

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A THEORETICAL METHOD FOR SELECTING
SPACE CRAFT AND SPACE SUIT ATMOSPHERES

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ABSTRACT

A theoretical method for selecting space craft and space suit atmospheres is described. The method assumes that gas bubbles cause decompression sickness and that the risk increases when a critical bubble volume is exceeded. The method is consistent with empirical decompression exposures for humans under conditions of nitrogen equlibrium between the lungs and tissues. Space station atmospheres are selected so that flight crews may decompress immediately from sealevel to station pressure without preoxygenation. Bubbles form as a result of this decompression but are less than the critical volume. The bubbles are absorbed during an equilibration period after which immediate transition to suit pressure is possible. Exercise after decompression and incomplete nitrogen equilibrium are shown to increase bubble size, and these factors limit the usefulness of one previously tested stage decompression procedure for the Shuttle. The method might be helpful for evaluating decompression procedures before testing.

decompression

decompression sickness

decompression theory

decompression limits

altitude bends

bubbles

space Shuttle

space station

The atmospheres used in the U.S. space program have been 100% oxygen at a pressure of about 5 psia in Gemini, Apollo, and Skylab (3) and air at sealevel pressure in the Shuttle. On the Apollo-Soyuz mission, the Russian atmosphere was 31% oxygen and 69% nitrogen at a pressure of 10 psia (6). Spacesuit atmospheres have been 100% oxygen at pressures of between 3.5 and 4.3 psia (1,3).

Many conflicting factors enter into the selection of these atmospheres. A pure oxygen atmosphere at low pressure allows considerable savings in vehicle weight and permits immediate transition of a flight crew to suit pressure for extravehicular activity (EVA) without the risk of decompression sickness (4). At low pressures, however, voice communications are difficult, and heat transfer efficiency is reduced which interfers with the cooling of electrical equipment. With pure oxygen, the fire hazard is increased, and there is a risk of aural and pulmonary atelectasis (17). These problems are avoided in the air atmosphere of the Shuttle, but a long decompression procedure is necessary before the crew can safely perform EVA at the present suit pressure of 4.3 psia (22). A higher pressure suit might eliminate the decompression requirement, but suit flexibility would be sacrificed.

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This paper describes a theoretical method for predicting those atmospheres which avoid the risk of decompression sickness. With this knowledge, the other factors affecting atmosphere selection can be evaluated independently. The method has evolved from the work of Nims (15), Hills (13), Hennessy and Hempleman (10), and Vann (21).

The method is most applicable to long duration missions such as might occur in a space station. In this situation, there would be adequate time after decompression from sealevel for nitrogen in the lungs and tissues to equilibrate before further decompression to suit pressure. The problem of nitrogen elimination by oxygen breathing at sealevel is addressed indirectly in an analysis of stage decompression procedures for the Shuttle.

THE CAUSE OF DECOMPRESSION SICKNESS

Animal experiments and autopsies of divers and caisson workers led Hill (12) to conclude that decompression sickness was caused by undissolved gas in the blood and tissues. The variety and severity of the symptoms depended upon the volume and location of the gas. Unconsciousness and death were often found to result from gas bubbles in the brain, dyspnea and chokes from gas in the pulmonary arteries, and paralysis from gas in the spinal cord. The etiology of the minor forms of decompression sickness was less clear because smaller volumes of gas were involved, but it seemed probable that muscle and joint pain were the result of bubbles in ligaments, fascia, periosteum, muscle spindles, and nerve sheaths.

Undissolved gas is still widely accepted as the initiating cause of decompression sickness, but the responses of the body to this gas are now recognized to be biochemical as well as mechanical. The details of the mechanisms by which gas bubbles cause decompression symptoms are not completely understood and will not be directly addressed here. Instead, it is assumed only that the risk of symptoms is high when a large volume of undissolved gas is present and, conversely, that the risk is low when little gas is present. Furthermore, as proposed by Hempleman (9), it is postulated that major symptoms will be rare if minor symptoms can be avoided consistently. With these assumptions, the problem becomes to find the smallest gas volume which results in decompression sickness and to relate this volume to the smallest pressure reductions known to cause symptoms in man.

THE CRITICAL BUBBLE VOLUME HYPOTHESIS

Figure 1 shows a tissue which is decompressed from a barometric pressure PB1 to a pressure PB2 where a bubble of critical volume forms. The critical bubble volume is defined as the largest volume which can be present without causing decompression sickness. If the critical bubble volume is designated as Vc, then a pressure reduction to less than PB2 causes the bubble to become larger than Vc, and the risk of decompression sickness exists.

A relationship between bubble volume and pressure reduction can be found in the following manner. It is assumed that diffusion within the tissue is instantaneous and that the pressure change is very

rapid. Thus, no nitrogen is lost during decompression, and the quantity of nitrogen dissolved in the tissue at PB1 is equal to the sum of the dissolved nitrogen at PB2 plus the nitrogen in the bubble. This is an expression of the law of conservation of mass.

The molar quantity of nitrogen dissolved at PB1 is

$$ND1 = S*Vt*(PtN2)1$$

where S is the nitrogen solubility in tissue in moles/ml tissue/psi, Vt is the tissue volume in ml, and (PtN2)1 is the tissue nitrogen tension in psi. Similarly, the molar quantity of dissolved nitrogen at PB2 is

$$ND2 = S*Vt*(PtN2)2$$

where (PtN2)2 is the nitrogen tissue tension at PB2 in psi. Although the tissue may have both fat and lean components, its nitrogen solubility is represented by a single quantity, S, as diffusion has been assumed to be instantaneous.

The molar quantity of nitrogen in the bubble at PB2 is defined by the ideal gas law as

$$NB2 = \frac{(PtN2)2*Vc}{RT}$$

where R and T are the general gas constant and the absolute temperature in appropriate units. The nitrogen partial pressure in the bubble and the tissue nitrogen tension are equal because of instantaneous diffusion.

By the law of conservation of mass, the dissolved nitrogen (ND1) at PB1 is equal to to the sum of the dissolved nitrogen (ND2) at PB2

and nitrogen in the bubble (NB3) at PB2 or

$$ND1 = ND2 + NB2$$

Substituting for these terms and solving for (PtN2)1 gives

$$(PtN2)1 = (Ac + 1)*(PtN2)2 \tag{1}$$

where

$$Ac = \frac{Vc}{S*Vt*R*T}$$

Ac is the ratio of the moles of nitrogen in the bubble to the moles of nitrogen dissolved in tissue.

At PB1, Dalton's law of partial pressures requires that the sum of the alveolar partial pressures of nitrogen, oxygen, and carbon dioxide (PAN2, PAO2, and PACO2) and the water vapor pressure (PH2O) be equal to PB1 or

$$PB1 = PAN2 + PAO2 + PACO2 + PH2O$$

If the alveolar partial pressures of all gases are equal to their corresponding arterial tensions (PaN2, PaO2, and PaCO2), then from above

$$PB1 = PaN2 + PaO2 + PaCO2 + PH2O$$

Since the tissues are assumed to be in equlibrium with nitrogen at PB1, the tissue nitrogen tension is equal to the arterial tension, and it can be found that

$$(PtN2)1 = PB1 - PaO2 - PaCO2 - PH2O \tag{2}$$

Rahn and Fenn (16) give the alveolar oxygen partial pressure as

$$PAO2 = PIO2 - PaCO2/RQ + FIO2*(PaCO2/RQ - PaCO2)$$

where PIO2, RQ, and FIO2 are the inspired oxygen partial pressure,

the respiratory quotient, and the inspired oxygen fraction. Setting

$$PAO2 = PaO2$$

and

$$PIO2 = FIO2*PB1$$

and letting

$$RQ = 1$$

which introduces little error, it is found that

$$PaO2 = FIO2*PB1 - PaCO2$$

Substituting this for PaO2 in (2), gives

$$(PtN2)1 = (1 - FIO2)*PB1 - PH2O \qquad (3)$$

In the bubble at PB2, Dalton's law requires that

$$PB2 + Pe = (PtN2)2 + PtO2 + PtCO2 + PH2O$$

where Pe is a pressure component due to surface tension and tissue elasticity and PtO2 and PtCO2 are the oxygen and carbon dioxide tensions in tissue. Solving for (PtN2)2,

$$(PtN2)2 = PB2 + Pe - PtO2 - PtCO2 - PH2O \qquad (4)$$

Substituting (3) and (4) for (PtN2)1 and (PtN2)2 in (1) and solving for PB2,

$$PB2 = \frac{(1 - FIO2)*PB1 - PH2O}{Ac + 1} - Pe + PtO2 + PtCO2 + PH2O \qquad (5)$$

MARGINAL DECOMPRESSION EXPOSURES

Equation (5) predicts the pressure PB2 to which decompression is marginally safe after an exposure at a pressure PB1 of sufficient length to ensure equlibrium between nitrogen in the lungs and in the tissues. If venous values are assigned to PtO2 and PtCO2, then

```
PtO2  = 40 mmHg for tissue oxygen tension,
PtCO2 = 45 mmHg for tissue carbon dioxide tension,
PH2O  = 46 mmHg for water vapor pressure, and
FIO2  = 0.21 for the oxygen fraction at PB1 if air is used.
```

The remaining unknowns, Ac and Pe, can be found if two pairs of marginal decompression exposures are known.

For the first marginal exposure, PB1 is sealevel pressure (14.7 psia), and PB2 is the lowest altitude at which decompression sickness occurs. Fryer (7) reports this altitude to be 18,500 ft. To be conservative, PB2 will be taken as a lower altitude of 18,000 ft (PB2 = 7.35 psia). For the second exposure, PB1 is the shallowest depth which results in decompression sickness when an air-equilibrated diver ascends directly to the surface (PB2 = 14.7 psia). Behnke and Jones (2) and Spencer (19) report this depth to be 25 feet of seawater (FSW). To be conservative, PB1 will be taken as 24 FSW (25.84 psia).

Substitution of the values

$$(PB1, PB2) = (14.7 \text{ psia, } 7.35 \text{ psia})$$

and

$$(PB1, PB2) = (25.84 \text{ psia, } 14.7 \text{ psia})$$

into equation (5) results in two equations with unknowns Ac and Pe which can be solved simultaneously to give

$$Ac = 0.1938$$

and

$$Pe = 4.1851 \text{ psi}$$

Since Ac is the ratio of nitrogen in the bubble to nitrogen dissolved in tissue, it can be found that about 16% of the total nitrogen is in

the bubble while 84% is dissolved. As the values for Ac and Pe were derived from the smallest pressure reductions reported to cause marginal decompression illness, it is expected that decompression procedures based upon them will be safe for a greater fraction of the population.

If Pe is assumed to be constant at all bubble volumes, then equation (5) holds for any bubble volume, V, where the parameter Ac is replaced by the parameter A defined as

$$A = \frac{V}{S*Vt*R*T}$$

Dividing A by Ac, it can be shown that

$$A = Ac*\frac{V}{Vc}$$

where a risk of decompression sickness exists if V/Vc exceeds 1.0. With this definition for A, equation (5) becomes

$$PB2 = \frac{(1 - FIO2)*PB1 - PH2O}{Ac*V/Vc + 1} - Pe + PtO2 + PtCO2 + PH2O \quad (6)$$

SPACE STATION ATMOSPHERES AND SUIT PRESSURES

Equation (6) may be used to find the final pressure PB2 which results in a bubble of fractional volume V/Vc after decompression from an initial pressure PB1 and oxygen fraction FIO2. Figure 2 illustrates how this equation might be applied to the transfer of a flight crew from sealevel to a space station where they would remain for an extended period performing regular EVA.

A Shuttle having an air atmosphere at 1 ATA transports the crew from Earth to an orbiting space station. Upon arrival at the station, the crew passes through an airlock into the station which has a pressure (PB)station and an oxygen fraction (FIO2)station. After sufficient time to permit nitrogen in their tissues to equilibrate with nitrogen in the station atmosphere, the crew decompresses to a pressure (PB)suit in 100% oxygen for EVA. Decompression to both (PB)station and (PB)suit results in the formation of a bubble of fractional volume V/Vc. (A similar procedure is used if the bubbles are of unequal volume.)

To find the station pressure, the desired bubble volume V/Vc is substituted into equation (6) where FIO2 is 0.21 and PB1 is 1 ATA. The suit pressure is determined in the same manner after substituting the calculated value of (PB)station for PB1 and the appropriate values for V/Vc and (FIO2)station. This procedure was used to develop Table 1 for V/Vc values of 0.0, 0.2, 0.4, 0.6, 0.8, and 1.0 and for (FIO2)station values of from 0.27 to 0.34. For example, if bubbles having a fractional volume of 0.4 are permitted to form and if the station FIO2 is 0.30, (PB)station is 8.3 psia and the lowest allowable (PB)suit is 2.91 psia. The oxygen partial pressures in the station and suit under these conditions are 0.17 and 0.20 ATM.

The atmospheres in Table 1 are subject to restrictions imposed by hypoxia, suit flexibility, fire hazard, and decompression risk. Resting men can tolerate oxygen partial pressures as low as 0.11 ATM with little difficulty, but their capacity for exercise is severely

limited (14). In experiments where men exercised on a bicycle ergometer while breathing air at simulated altitudes, decreased exercise tolerance was observed at 10,000 feet but not at 5,000 feet (20). The oxygen partial pressure in air at 5,000 feet is about 0.17 ATM (2.5 psi), and this value has been applied to Table 1 as the lower limit for hypoxic exposure.

Another limitation to exercise capacity is the stiffness of the suits which flight crews wear during EVA. These suits are most flexibile at low pressures. Since the gas in them is 100% oxygen, the lowest possible suit pressure is set by the hypoxic limit of 0.17 ATM (2.5 psi). Suit pressures above the current level of 4.3 psia could be used if a more flexible design were available.

If the station PIO_2 is not to be less than the hypoxic limit when the station pressure is below 12 psia, the FIO_2 must be raised above 0.21. Associated with this rise is an increase in fire hazard which can be measured as a percentage decrease in combustion time relative to the combustion time in air at 14.7 psia. Estimates of decreased combustion times for elevated FIO_2's were derived from data of Simons and Archibald (18). These estimates are listed in Table 1 and show that as the FIO_2 rises from 0.21 to 0.34, the combustion time decreases by approximately 24%.

Table 1 can be thought of as a risk-benefit matrix in which the station atmosphere is determined by the station FIO_2, and this is chosen in a compromise between suit flexibility, fire safety, and decompression risk. The risk of hypoxia increases diagonally from the

bottom left corner to the top right corner. Lower suit pressures and greater suit flexibility are achieved by moving in the same direction. Fire hazard increases from top to bottom as the station FIO_2 rises. The risk of decompression sickness increases when moving from left to right as the fractional bubble volume increases from 0.0 to 1.0. Atmospheres having the larger bubble volumes should be avoided since exercise after decompression causes an increased risk of decompression sickness (8).

STAGE DECOMPRESSION PROCEDURES FOR THE SHUTTLE

The effect of exercise after decompression can be quite significant. Gray found that the incidence of decompression sickness at pressures of 3.8 to 3.0 psia was increased 15 to 32% in subjects who did 5 push-ups and 5 deep knee bends every 15 minutes (5). The increased incidence was approximately equivalent to an extra 5,000 feet of decompression.

Henry observed that the rise in decompression sickness was related to the severity of the exercise, and he concluded that locally elevated CO_2 production was causing bubbles to expand (11). Burkhardt, on the other hand, found that the incidence was reduced by hyperventilation which Nims concluded was an indication of a decrease in bubble volume as a result of CO_2 elimination (15).

These explanations suggest that exercise might be simulated by a virtual decompression which causes existing bubbles to expand. This tactic can be used to analyze the effects that exercise may have had

during tests of stage decompression procedures for the Shuttle.

Adams et al. (1) exposed 18 subjects to 9.2 psia on 28% oxygen for 12 hours and then on 100% oxygen for 45 minutes before ascent to a pressure of 4.0 psia. After decompression, the subjects exercised for 1 out of every 8 minutes. This procedure resulted in one case of decompression sickness. In another test, Waligora et al. (22) exposed 50 subjects to 10.2 psia on 26% oxygen for 12 to 18 hours and then on 100% oxygen for 40 to 90 minutes before ascent to 4.3 psia. Upon arrival at 4.3 psia, the subjects exercised for 8 out of every 16 minutes. This procecure resulted in 15 cases of decompression sickness.

It is uncertain whether these procedures produced complete equilibrium between nitrogen in the lungs and tissues. In the event that equilibrium was not complete, $(PtN2)1$ is given by

$$(PtN2)1 = Feq*((1 - FIO2)*PB1 - 0.79) + 0.79 - PH2O \qquad (7)$$

where Feq is the nitrogen equilibrium fraction. Before any equilibration has occurred, Feq is zero and

$$(PtN2)1 = 0.79 - PH2O$$

After equilibrium is complete, Feq is one and $(PtN2)1$ is given by equation (3).

With the aid of equation (7), it may be found that

$$\frac{V}{Vc} = (\frac{Feq*((1 - FIO2)*PB1 - 0.79) + 0.79 - PH2O}{PB2 + Pe - PtO2 - PtCO2 - PH2O} - 1)/Ac \qquad (8)$$

Equation (8) was used to generate Figs. 3 and 4 which show the effects of exercise and incomplete nitrogen equilibrium on the 9.2

and 10.2 psia Shuttle decompression procedures. Exercise is simulated by a virtual decompression of 5,000 ft as suggested by Gray's observations (5). This is probably too great for the 9.2 psia procedure but may be reasonable for the 10.2 psia procedure.

The 9.2 psia procedure had a 6% incidence of decompression sickness. In Fig. 3, this procedure is seen to be safe (V/Vc < 1.0) for resting subjects when nitrogen equilibrium is more than 80% complete. For exercising subjects, however, it is safe only at complete equilibrium. Nitrogen equlibrium is estimated to have been 80 to 90% complete.

The 10.2 psia procedure had a decompression sickness incidence of 30%. In Fig. 4, this procedure is seen to be safe for resting subjects when nitrogen equilibrium is more than 90% complete. The procedure appears to be unsafe, however, for anymore than minimal exercise.

This discussion has illustrated a retrospective use of equation (8). A similar analysis applied in advance might aid the selection of space craft atmospheres and the development of stage decompression procedures for the future.

ACKNOWLEDGMENTS

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Table 1. Station and suit atmospheres. (PIO2 is in ATM and PB is in psia.)

| (FIO2) station	V/Vc	0.0	0.2	0.4	0.6	0.8	1.0	% Decrease in Combustion Time
	(PB)station	9.07	8.67	8.30	7.95	7.63	7.33	
0.27	(PIO2)station	0.17	a	a	a	a	a	14
	(PB)suit	4.08						
	(PIO2)suit	0.28						
0.28	(PIO2)station	0.17	a	a	a	a	a	15
	(PB)suit	3.99						
	(PIO2)suit	0.27						
0.29	(PIO2)station	0.18	0.17	a	a	a	a	17
	(PB)suit	3.90	3.42					
	(PIO2)suit	0.27	0.23					
0.30	(PIO2)station	0.19	0.18	0.17	a	a	a	19
	(PB)suit	3.81	3.34	2.91				
	(PIO2)suit	0.26	0.23	0.20				
0.31	(PIO2)station	0.19	0.18	0.18	0.17	a	a	21
	(PB)suit	3.72	3.25	2.84	2.47			
	(PIO2)suit	0.25	0.22	0.19	0.17			
0.32	(PIO2)station	0.20	0.19	0.18	0.17	0.17	a	22
	(PB)suit	3.63	3.17	2.76	2.40	2.50		
	(PIO2)suit	0.25	0.22	0.19	0.16	0.17b		
0.33	(PIO2)station	0.20	0.19	0.19	0.18	0.17	a	23
	(PB)suit	3.54	3.08	2.68	2.50	2.50		
	(PIO2)suit	0.24	0.21	0.18	0.17b	0.17b		
0.34	(PIO2)station	0.21	0.20	0.19	0.18	0.18	0.17	24
	(PB)suit	3.45	3.00	2.61	2.50	2.50	2.50	
	(PIO2)suit	0.23	0.20	0.18	0.17b	0.17b	0.17b	

a — (PIO2)station < 0.17 ATM
b — (PIO2)suit raised to 0.17 ATM

FIGURE CAPTIONS

1. Formation of a bubble of critical volume.

2. Decompression from sealevel to station and suit pressures.

3. Shuttle decompression procedure with PB1 = 9.2 psia, FIO2 = 0.28, and PB2 = 4.0 psia. Decompression sickness incidence was 6%.

4. Shuttle decompression procedure with PB1 = 10.2 psia, FIO2 = 0.26, and PB2 = 4.3 psia. Decompression sickness incidence was 30%.
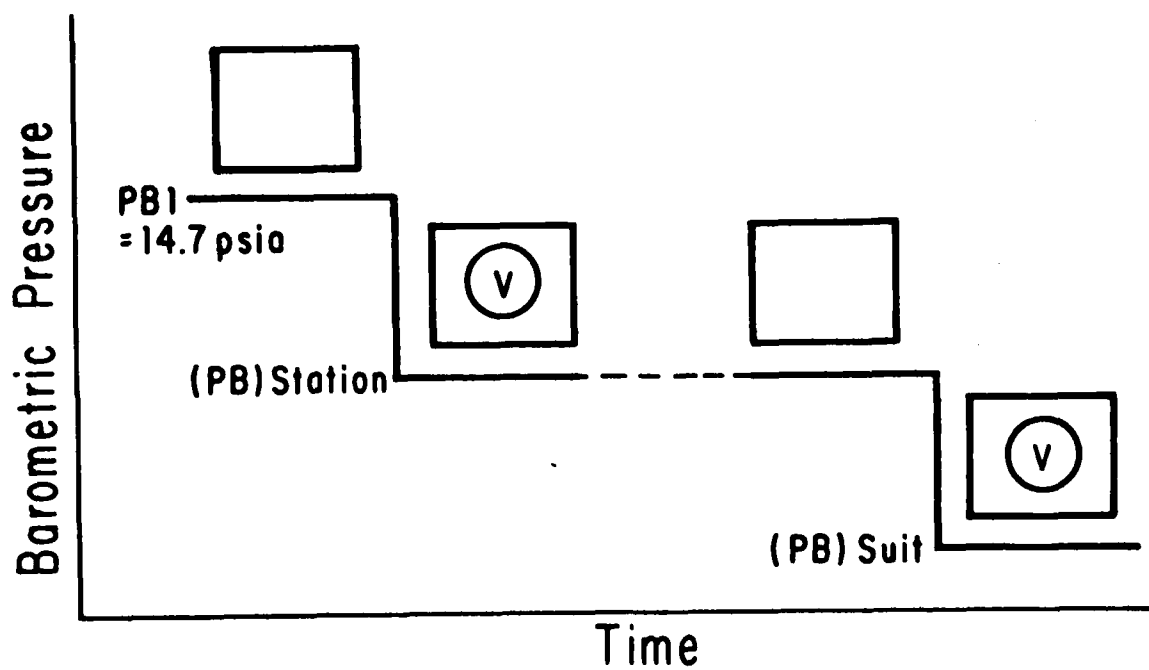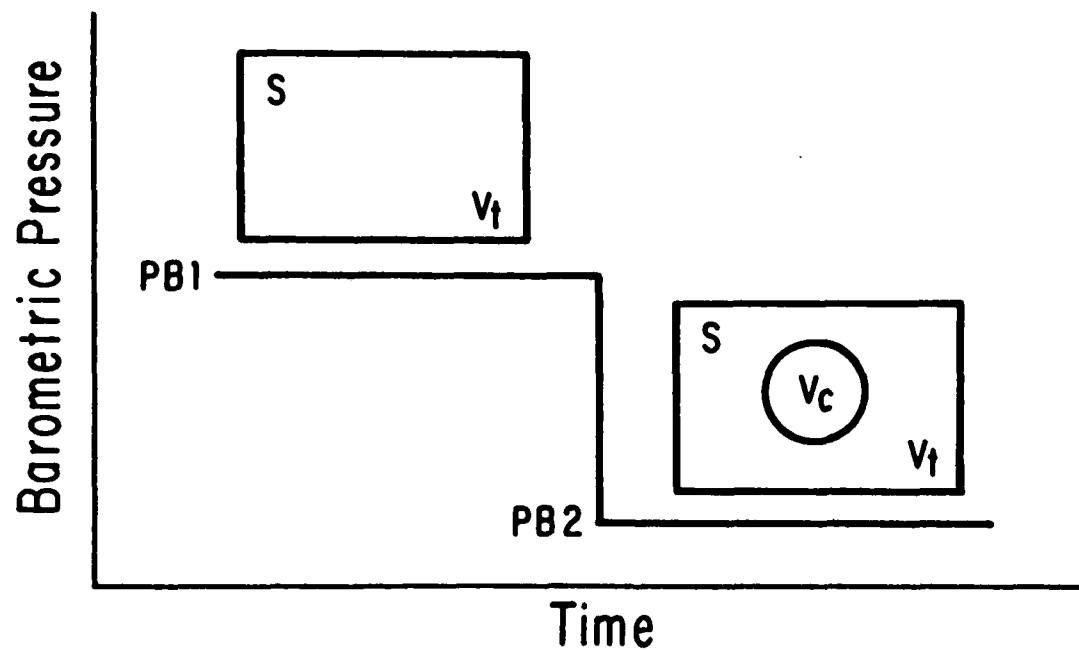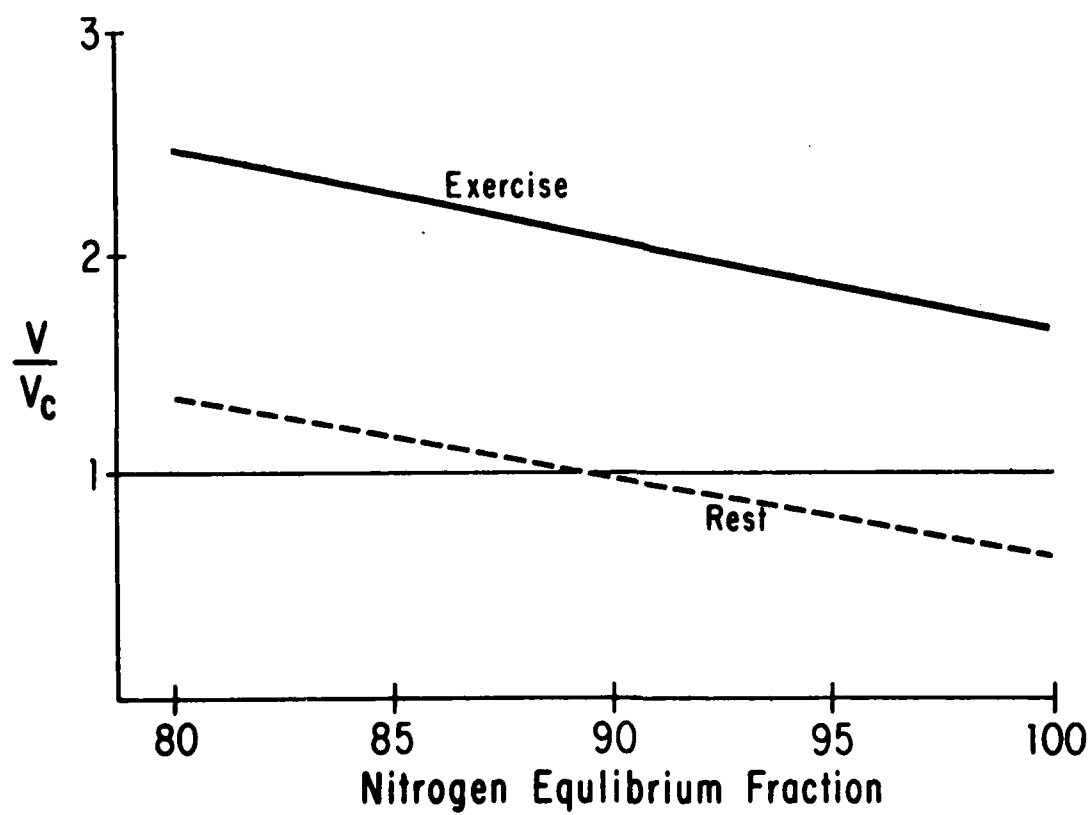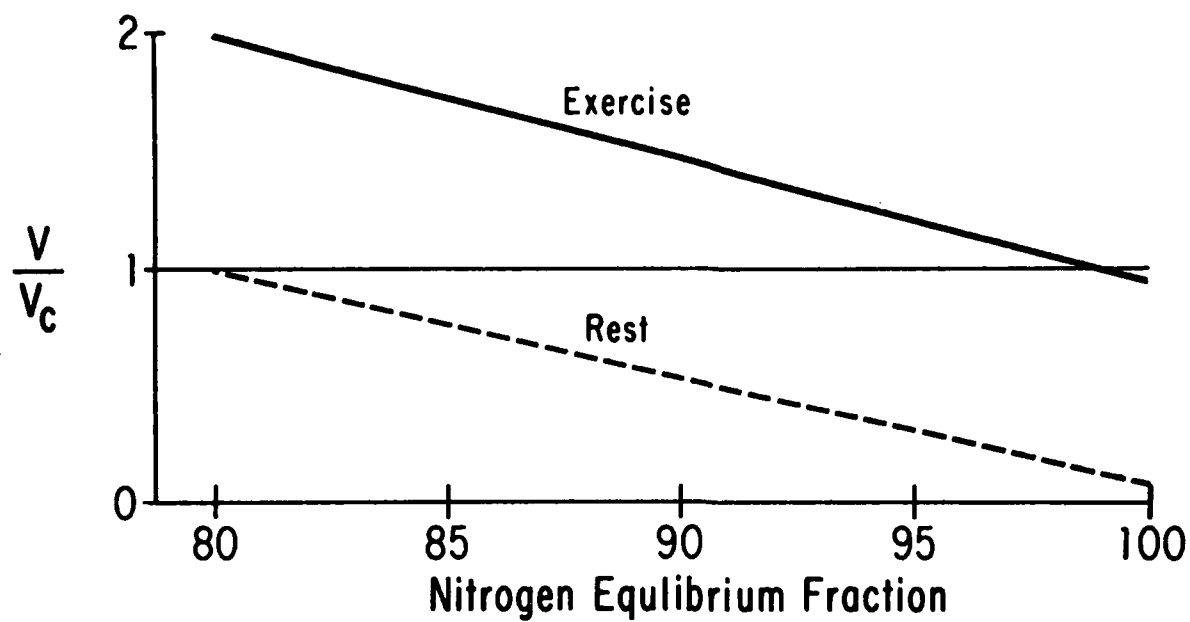
DECOMPRESSION FROM A DEEP NITROGEN/OXYGEN SATURATION DIVE

A CASE REPORT

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Running Head: Nitrogen/Oxygen Saturation Decompression

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ABSTRACT

Ten divers participated in a 4.5 day nitrogen/oxygen saturation dive to 165 fsw. There were daily 2 hour excursions to 200 fsw. The divers breathed air during the excursions and 0.5 ATM oxygen in nitrogen at 165 fsw. The final decompression began 6 hours after the last excursion. The oxygen partial pressure was 0.5 ATM from 165 to 45 fsw, and air was used from 45 fsw to the surface. By 20 fsw, 4 divers had developed decompression sickness. A fifth diver developed decompression sickness during a commercial air flight 68 hours after surfacing. Comparison of ascent rates for this dive and for air or nitrogen/oxygen saturation dives reported in the literature suggests that deeper dives require slower rates of ascent. Dives shallower than 100 fsw had a mean ascent rate of 3.2 fsw/hr and 14 decompression incidents in 107 man-exposures. Dives deeper than 100 fsw had a mean rate of 2.5 fsw/hr and 14 incidents in 45 man-exposures.

decompression sickness

saturation diving

nitrogen/oxygen

flying after diving

ascent rates

To determine if man could adapt to nitrogen narcosis, Oceaneering International, Inc. and the National Oceanic and Atmospheric Administration sponsored a nitrogen/oxygen saturation dive in the chambers of the F. G. Hall Laboratory at Duke University Medical Center. This communication describes and discusses the clinical results of the decompression from this dive.

Few studies describing decompression after nitrogen/oxygen saturation diving have been reported (1-5), but this information is of major importance for the development of safe decompression procedures. It is hoped that publication of these data will encourage similar reports of decompression studies from other laboratories.

METHODS

Ten experienced and physically qualified divers (ages 22-42) were compressed at a rate of 60 ft/min in air to a simulated depth of 200 fsw. After two hours at this depth, the divers were decompressed in two minutes to 165 fsw and transferred to another chamber containing 0.5 ATM oxygen in nitrogen. The nitrogen partial pressure in the storage chamber at 165 fsw was equal to that of air at 200 fsw. During the next four days, the divers were compressed on air once a day to 200 fsw for two hours and then decompressed to 165 fsw. After 4.5 days, they were decompressed to the surface in approximately 3.5 days. Figure 1 illustrates the dive profile.

Performance and psychological studies were conducted pre-dive, at 165 and 200 fsw, during decompression, and post-dive, but no physical exercise took place. The divers completed questionnaires twice daily to assess their physical and mental well-being. The questions related

to the signs and symptoms of nitrogen narcosis, decompression sickness, and general health. The divers also kept daily logs for post-dive evaluation.

The final decompression began six hours after completion of the last excursion. The proposed decompression schedule is listed in Table 1. Travel time was approximately one minute between stops and was included in the time at the next stop. An oxygen partial pressure of 0.5 ATM was maintained until 45 fsw after which an oxygen percentage of 21% was maintained to the surface.

During decompression, the divers were encouraged to ingest large amounts of fluid to maintain a urine specific gravity of 1.004. Each diver measured his own specific gravity with a hydrometer. The temperature in the chamber was 82-84 degrees F during the day but was raised to 88-90 degrees at night to enhance perfusion. Some divers found these higher temperatures uncomfortable.

Precordial doppler bubble-detection studies were conducted at the 30, 25, 20, and 10 fsw decompression stops and shortly after arrival at the surface. To take doppler readings, the probe was positioned on the chest of a supine diver, and the diver was asked to flex each limb separately. Bubble scores were assigned according to the scale of Spencer (6). All except the post-dive readings were taken in the chamber.

RESULTS

Decompression was uneventful until 65 fsw where one diver complained of a vague, minor pain in his right ankle. This pain

occurred intermittently and became stronger as the 20 fsw stop was approached. A second diver reported a sharp pain in the left elbow while traveliing from 25 to 20 fsw. The pain lasted 1-2 minutes and subsided spontaneously. He also noted mild discomfort in both knees where he previously had a bilateral ilio-tibial band syndrome. This condition had developed six months before the dive but had been asymptomatic for two months. A third diver experienced transient, mild to moderate right elbow pain at 65 fsw and slight left knee pain during the move from 25 to 20 fsw.

A fourth diver, the youngest of the ten, developed fairly severe and constant pain in his right knee shortly before leaving the 30 fsw stop. The pain became worse until well into the 20 fsw stop where he reported tingling in the right calf, pain in the right leg when walking, and a steady, aching pain behind the right knee and in the right thigh. Neurologic examination by a diving medical officer, who was one of the divers, showed no objective signs of spinal cord involvement.

The four divers who had symptoms of decompression sickness were placed on 100% oxygen and compressed to 60 fsw for treatment on a modified U.S. Navy Treatment Table 6. This table was extended at 60 fsw to include one extra 20 minute oxygen cycle. Six oxygen cycles were given at 30 fsw. After treatment, the divers were returned to 20 fsw rather than to the surface.

While all divers had 100% relief of symptoms, the diver with the most severe symptoms experienced aggravation of his knee and thigh pain during the first oxygen cycle. He was 90% asymptomatic after the

third cycle and completely asymptomatic after the forth cycle. This
diver became dizzy during the ascent from 60 to 30 fsw, and all
divers were taken off oxygen for 15 minutes at 45 fsw for
observation. After the dizziness cleared and no other symptoms were
reported, decompression to 30 fsw was resumed.

The six divers who had not reported symptoms remained at 20 fsw
and breathed 100% oxygen for 25 minutes as a precautionary measure.
When the four treated divers arrived at 20 fsw, all divers received
five grains of aspirin and again were encouraged to remain well
hydrated. Decompression was resumed on the original schedule (Table
1) with two prophylactic oxygen cycles at 10 fsw and with oxygen
breathing during the 10 minute ascent from 10 fsw to the surface.

A fifth diver developed decompression sickness during a
commercial flight in a pressurized aircraft 68 hours after leaving
the chamber. His symptoms were severe pain in the knees, hips, back,
shoulders, elbows, wrists, and jaw. These symptoms persisted after
landing. Although he had reported no symptoms during the dive, his
log contained a record of mild, transient muscle pain in the left
deltoid and biceps area at a depth of 55-50 fsw. He had no other
aches or pains during or immediately after the dive. He was not one
of the divers treated on the modified Table 6 but because of nausea,
did not complete the precautionary oxygen-breathing period given the
other divers at 20 fsw.

During the 10 day period following his flight, he received one
U.S. Navy Treatment Table 6, four Treatment Table 5's, and one short
oxygen-breathing period at 30 fsw. Pulmonary oxygen toxicity
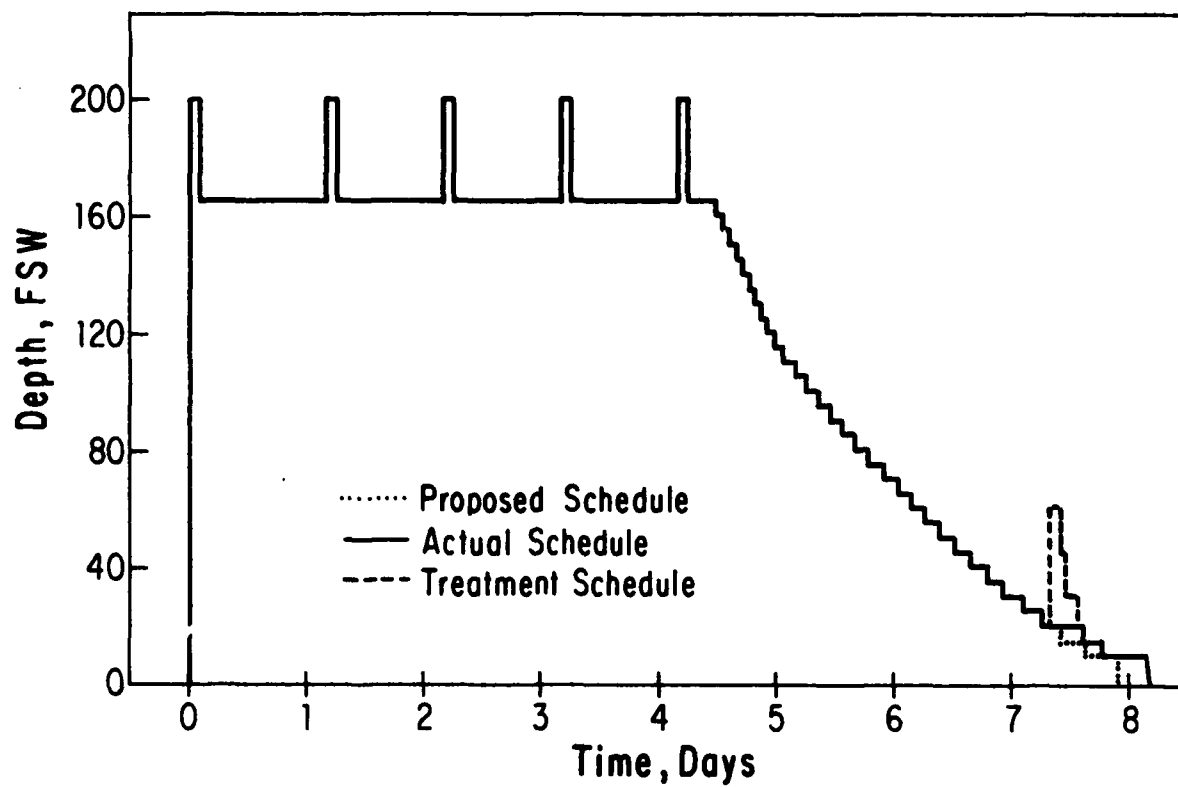
precluded further hyperbaric oxygen therapy. He also received anti-inflammatory agents and corticosteroids. After several months, he still reported occasional mild joint pain.

All divers had evidence of intravascular bubbles with doppler bubble grades ranging from 0 to 4, but eight had grades of no higher than 2. The diver who experienced left elbow pain when travelling from 25 to 20 fsw had a grade of 3 in his left arm at the 20 fsw stop. The diver who developed frank decompression sickness at 20 fsw had a grade of 4. In all divers with symptoms, the highest bubble grade was associated with the painful limb. Table 2 lists the bubble grades for both treated and untreated groups. While the difference in mean bubble grade between the two groups was not statistically significant, the treated divers tended to have higher bubble grades before recompression. The data are presented here to encourage publication of doppler measurements from other saturation dives. Statistically significant trends may become evident if enough data are available.

DISCUSSION

The ascent from this unusually deep nitrogen/oxygen saturation dive was uneventful until 65 fsw after which 5 of the 10 divers developed symptoms of decompression sickness. The average rate of ascent from 165 to 65 fsw was 2.7 fsw/hr (2.2 fsw/hr from 165 to 20 fsw). Table 3 shows the average ascent rates until the depth of the first symptoms for this dive and for other air or nitrogen/oxygen saturation dives (1-5). For dives to less than 100 fsw, the incidence of decompression sickness was 13.1% (14 incidents in 107

man-exposures), and the mean ascent rate was 3.2 fsw/hr. For dives deeper than 100 fsw, the incidence was 31.1% (14 incidents in 45 man-exposures), and the mean ascent rate was 2.5 fsw/hr. The higher decompression sickness incidence and the lower ascent rate for the deeper dives suggest that slower rates of ascent are necessary for deeper dives. Muren et al. (4) propose a similar hypothesis.

It can be found from Table 1 that the ascent rate from 165 to 115 fsw was 3.8 fsw/hr while the rate from 115 to 65 fsw was 1.9 fsw/hr. The shallower rate is slow compared to the rates in Table 3 suggesting that the faster initial rate may have been responsible for subsequent decompression sickness.

Other nitrogen/oxygen saturation dives must be conducted and their results published if the effect of depth on ascent rate is to be resolved and if satisfactory decompression schedules are to be developed.

ACKNOWLEDGEMENTS

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Table 1. Decompression schedule.

DEPTH (FSW)	STOP TIME (MIN)
165-115	80 (at each 5 fsw stop)
115	90
110	140
105	145
100	145
95	150
90	150
85	150
80	150
75	155
70	160
65	160
60	165
55	190
50	190
45	195
40	195
35	215
30	215
25	230
20	230
15	245
10	525
10-0	10

Table 2. Doppler bubble grades.

MAXIMUM BUBBLE GRADE

		2	3	4
	30	3(3)*	(3)	1
	25	2(4)	1(2)	1
DEPTH (FSW)	20	2(6)	1	1
	10	2(2)	2(4)	
	0	(1)	4(5)	

* Number of treated divers. (Number of untreated divers are shown in parenthesis.)

Table 3. Average ascent rates until the occurrence of
decompression sickness for published air and nitrogen/oxygen
saturation dives.

DIVE NAME (REFERENCE)	SATURATION DEPTH (FSW)	RATE (FSW/HR)	% DCS (NO. DCS/ NO. DIVERS)
Pre-SHAD (2)	50	7.60	100 (2/2)
SHAD 1 (2)	50	3.70	0 (0/2)
SHAD 3 (2)	50	3.68	33 (1/3)
SHAD 2 (2)	60	3.74	50 (1/2)
SCORE (1,5)	60	3.48	15 (7/48)
AIRSAT 1 & 2 (3)	60	3.28	9 (2/23)
SUREX 65 (3)	65	2.10	6 (1/18)
SUREX 75 (3)	75	2.18	0 (0/6)
NOAA OPS 1 (1)	90	3.91	0 (0/3)
AIRSAT 3 (3)	132	2.55	25 (3/12)
AIRSAT 4 (3)	132	2.18	7 (1/15)
OI/NOAA/DUKE	165	2.69	50 (5/10)
NISAT I (2)	198	1.23	0 (0/2)
NISAHEX (4)	198	2.92	83 (5/6)

FIGURE CAPTION

1. Dive profile. The breathing gas was 0.5 ATM oxygen in nitrogen at 165 fsw and air at 200 fsw. During decompression, the oxygen partial pressure was 0.5 ATM from 165 to 45 fsw. Air was used from 45 fsw to the surface.

AIR AND NITROX SATURATION DECOMPRESSION:

A report of 4 schedules and 74 subjects

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RUNNING HEAD: Nitrox saturation decompression

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ABSTRACT

Seventy-four subjects were decompressed from air or
nitrogen-oxygen saturation exposures at 60 to 132 feet sea water
gauge (fswg) using four different decompression schedules. A
twenty hour schedule for decompression from an air
saturation-excursion profile at 60 fswg resulted in pain-only
decompression sickness (DCS) symptoms in two of twenty-three
subjects. A thirty-two hour schedule from a different air
saturation profile at 65 and 75 fswg resulted in DCS symptoms in
one of twenty-four subjects. A third and fourth schedule for air
or nitrox saturation at 132 fswg resulted in DCS symptoms in
three of twelve and one of fifteen respectively. No serious (type
II) symptoms were observed as a result of any of the exposures.
All cases consisted of knee pain occurring either in the last 10
fsw of the decompression, or shortly after surfacing. Doppler
ultrasound monitoring revealed venous gas emboli (VGE) in several
subjects, but generally only shallow to 20 fswg. Results
demonstrate an overall DCS incidence of 9.5%, and all cases were
pain-only in character. Thus, the timing and character of DCS
from air and nitrogen-oxygen saturation exposures is similar to
that of helium exposures. Although differentiation between the
presented schedules is impossible due to the limited number of
subjects in each, the results suggest that the shallow ascent
rate is more important in determining DCS incidence than that
from deeper portions of the schedule.

INDEX TERMS: diving; humans; decompression; inert gas; saturation

INTRODUCTION

A confusing array of decompression schedules for saturation on air at increased ambient pressure has been formulated and tested over the past twenty years (1-9). The incidence of decompression sickness (DCS) has varied widely depending on individual susceptibility and characteristics of the particular exposure. Attempts to apply existing data to decompression schedule formulation have been complicated by different levels of exercise, temperature, carbon dioxide, oxygen and wet versus dry environments. Moreover, since much decompression information never reaches publication, subsequent analysis must rely on hearsay or inadequate data. To formulate sound schedules, and more thoroughly understand the physiology of saturation decompression, a large number of decompression trials with a variety of environmental conditions is required. To this end, this report is a detailed description of the conditions and results of air saturation decompressions occurring at this laboratory in the course of experiments over the past six years. This is not intended to be a comprehensive investigation into the theory and physiology of air saturation decompression, nor is it intended to promote the use of the described schedules. Rather, it is intended to add to the body of knowledge of how humans tolerate decompression from air saturation under a variety of conditions.

MATERIALS AND METHODS

Subjects

The subjects for these exposures were active duty or reserve
Navy divers, with varying degrees of diving experience, and all
of whom had been in hyperbaric chambers previously. No subject
had been exposed to elevated pressure for at least two weeks
preceeding the saturation exposure. Subject vital statistics for
each series of experiments are shown in Table 1. No significant
differences existed between the groups of subjects, with the
exception that the AIRSAT 3 subject population was significantly
older than that in AIRSAT 2. Informed consent was obtained prior
to any exposures or procedures.

Facility

All saturation exposures were performed in the main hyperbaric
chamber of the Environmental Simulation Facility located at the
Naval Submarine Medical Research Laboratory in Groton, Ct. The
chamber was of double lock design, steel construction and was man
certified to 350 fswg. The diameter measured 9 feet, the inner
lock being 15 feet in length and the outer 10 feet. Separate life
support systems for each lock controlled CO_2, temperaure and
humidity. CO_2 was monitored continuously in each lock by Beckman
864 analyzers and averaged $0.06 \pm .02\%$ for all exposures. Oxygen

was monitored by Beckman 755 analyzers and was maintained at plus or minus 1% of the desired value by Teledyne 323 controllers. For clarification, the oxygen partial pressure profile for each of the exposures is shown in Fig.2. Temperature was adjusted to subject comfort and averaged 25.8 $\pm$ 0.9 degrees Celcius. All travel during decompression occurred in 1 fsw increments. Diet was not controlled or limited. Sleeping habits were generally not altered. Medications were discouraged and rarely used. Occasionally, acetaminophen, topical antifungal preparations, antacids, pseudoephedrine and topical decongestants were used. Analgesics were not administered during decompression. Activity levels generally consisted of unhurried movement about the chamber (exercise studies discussed below). Scientific procedures generally consisted of spirometry, other non-strenuous breathing tests, blood draws, special sensory tests (vision, hearing), psychomotor tests, EEGs, ECGs and the like. No oxygen breathing was included in any of the decompression schedules.

Decompression monitoring

Adequacy of decompression was gauged primarily by reported and elicited symptomatology. Symptoms were divided into conventional categories: pain only symptoms (type I) and multisystem or serious symptoms (type II), and treated according to guidelines detailed in the Navy Diving Manual (10). Occasionally the oxygen dose was modified when treating a subject with significant symptoms of pulmonary oxygen toxicity as a result of the

saturation exposure. Precordial monitoring for right heart venous gas emboli (VGE) using doppler ultrasound was carried out at regular intervals during the decompression in schedules 2 and 4 only. A Sodelec D.U.G. unit with probe was used for the signal generation, and the Kisman-Masurel scoring scheme (11) was used for analysis of the signals. In this system, two scores are reported - one representing the VGE score with the subject standing at rest, and the other after a series of three deep knee bends (rest/movement).

SCHEDULE DESCRIPTION AND RESULTS

Schedule #1

The decompression schedule is shown in Table 3[1]. This schedule is based on the NOAA 3202 M-value matrix, assuming that the 480 minute half time tissue is the rate limiting compartment (2). This decompression was used for two series of air saturation exposures (AIRSAT 1 and 2), with a total of 23 subjects. The storage depth was 60 fswg, and daily excursions to 100 fswg or 150 fswg were made as shown in Fig.1A & B. Fifteen hours and 45 minutes elapsed between the 100 fswg excursions and 19 hrs and 20 min between the 150 fswg excursions. Each subject exercised on a bicycle ergometer for 30 minutes at approximately 75% of maximal capacity - pre-dive, on the excursions only during the dive, and post-dive. The final decompression began about 44 hours after the final excursion in AIRSAT 1 and 47 hours after the final excursion in AIRSAT 2. No symptoms resulted from any of the excursions in these experiments.

Twent·-three subjects decompressed from the above profiles using this schedule. One subject (AIRSAT 1) was classified as having type I decompression sickness. Briefly, this subject noted mild, deep seated right knee discomfort on awakening at 6 fswg, which then increased to moderate levels on reaching the surface. A similar pain had begun to appear in the left knee by the time

recompression therapy was initiated (about 30 minutes after surfacing). Complete relief of the left knee discomfort and 90% relief of the right knee pain was achieved in the first 10 minutes of treatment. To minimize further oxygen stress, the second oxygen period at the 60 fsw level was eliminated, and the remainder of a standard U.S.Navy treatment table 5 (TT5) was completed. Complete relief of all symptoms with no recurrence was the final outcome.

One other subject (AIRSAT 2) reported a vague feeling of discomfort in the left knee about 3 hours after reaching the surface, but it resolved after several hours with no treatment. None of the other 21 subjects reported symptoms during or subsequent to the decompression. Doppler monitoring was not performed during this series.

Schedule #2

Schedule #2 was developed using the empirical relationship

$$R = k(PiO_2) \qquad (1)$$

where R is the rate of ascent in fsw/hr, PiO_2 is the inspired oxygen partial pressure in ATA, and k is 6. Since the breathing media for this schedule was air, the PiO_2 decreased as the depth decreased. To satisfy equation (1), the ascent rate also was

reduced. For convenience, the ascent rate was reduced at 10 fsw intervals to the rate required by the lowest PiO_2 in the interval.

Schedule #2 was used for a series of eight exposures (the SUREX experiments), each with three subjects. The atmosphere was air throughout, and excursions to the surface were included (see Fig.1C & D). Further details of this exposure is contained in the references (12). The subjects spent a total of 44 hours at 65 fswg (SUREX 1-6), or 75 fswg (SUREX 7,8), and the decompression began about 20 hours after completion of the final ascending excursion. The excursions represented a significant decompression stress, and most subjects complained of pruritus and had detectable VGE during the surface interval. Four subjects had DCS symptoms (3 type I and 1 type II) during or immediately following the excursions. All subjects were asymptomatic prior to initiating the final decompression.

One of the twenty-four subjects (not one of the four with DCS symptoms during the excursions) noticed mild, deep seated left knee pain at about 2 fswg, which was essentially unchanged on arrival at the surface. Physical exam was entirely normal. A standard TT6 was initiated, and full relief was obtained after the first oxygen breathing period at 60 fswg. There was no recurrence.

The number of subjects with detectable VGE and the mean VGE

score are shown in Table 3. In this schedule, no VGE were detected deeper than 10 fswg. The highest VGE score occurred in the subject with diagnosed decompression sickness (rest grade 2/movement grade 4). The other scores were generally very low.

Schedule #3

This decompression schedule used is the U.S. Navy standard helium-oxygen saturation decompression schedule (10). Because of the very low incidence of decompression sickness associated with the use of this schedule for shallow heliox exposures, it was believed that it might be sufficiently conservative to allow safe decompression from shallow air saturation exposures. The ascent rates are shown in Table 2. Rest stops are an integral part of this decompression. The protocol calls for rest stops (no travel) from 2400-0600 and from 1400-1600 independent of the starting time. Continuous travel occurs at all other times of the day.

This decompression was used exactly as indicated in a series of 4 identical air and nitrox saturation exposures (AIRSAT 3) each with three subjects. The pressurization and atmosphere profile is shown in Fig.1E. Briefly, daily 5 hour no-decompression excursions on air to 198 fswg (7 ATA) were made from a nitrogen-oxygen (PiO_2=0.30 ATA) storage depth of 132 fswg (5 ATA) on days 2,3 and 4. Eighteen hours and 45 minutes elapsed between the excursions. Twenty hours after the third and final excursion,

and isobaric shift to air occurred. The air exposure at 5 ATA continued for 24 hours at which time the final decompression began (at 1000 on day 6). The chamber reached the surface at 1346 on day 8.

Three of the 12 subjects were classified as having type I DCS. All three had left knee discomfort, but the time of onset was somewhat different. Two of the three subjects initially noted symptoms at about the 5 fswg level, and the other subject noticed symptoms about 3 hours after surfacing. One of the two subjects noting symptoms while still under pressure was treated according to the minimal decompression concept (10) i.e.; recompressed 10 fswg deeper than where symptoms were noted, held for two hours while breathing 100% oxygen, and then resumed the decompression schedule. Full relief was obtained in this subject. The other subject with symptoms under pressure was allowed to surface (he did not mention symptoms until this time), and then treated with a modified TT5[2]. This subject had complete relief 5 minutes into the first oxygen period at 60 fswg, but he had a recurrence of the knee pain only thirty minutes after surfacing from the treatment. A modified TT6 then resulted in complete relief with no recurrence, and surprisingly, no further pulmonary symptomatology or decrement in pulmonary function. The subject with symptoms after surfacing was treated with a modified TT6, and had complete relief with no recurrence. Doppler monitoring was not performed during these exposures.

Schedule #4

This schedule was also derived using equation (1), with k equal to 5 instead of 6. This resulted in slower ascent rates, which were believed to be prudent as the subjects were expected to develop symptoms of pulmonary oxygen toxicity (see Fig. 2C). Animal studies have suggested that pulmonary oxygen toxicity reduces decompression tolerance (13). Slower ascent rates also appear to be necessary for deeper saturation exposures. This is discussed below.

The exposure for which this schedule was used (AIRSAT 4) is shown in Fig.1F. Briefly, compression to 132 fswg (5 ATA) on an atmosphere of nitrogen-oxygen ($PiO_2 = 0.30$ ATA) is followed, 12 hours later, by an isobaric shift to air ($PiO_2 = 1.05$ ATA). No excursions were performed. Forty-eight hours after the isobaric shift, another isobaric shift back to nitrox occurred (now $PiO_2 = 0.50$ ATA), and the decompression started immediately (at 2200 on day 3). The partial pressure of oxygen was maintained at 0.50 ATA until the chamber oxygen level reached 21% (at 46 fswg), afterwhich, the FiO_2 of 21% was maintained to the surface (decreasing PiO_2). The chamber reached the surface at 1508 on day 6.

Fifteen subjects have decompressed from the above profile using this schedule. One subject had onset of bilateral knee discomfort (left greater than right) on awakening on day 6 at about 10 fswg.

This discomfort would generally abate before the pressure was again reduced by 1 fsw (every 58 minutes at this point), at which time it would re-appear. This continued to about 2 fswg, where he was transferred to another chamber and treated with a standard USN TT5. He had rapid resolution of the pain at 60 fswg on oxygen, and no recurrence. The other subjects in the chamber were allowed to continue the schedule. The symptomatic subject had the highest VGE score of the three (rest-1/movement-3), but VGE were undetectable after completion of the TT5. The number of subjects with detectable VGE and the mean VGE scores are shown in Table 3 below. VGE were detected deeper in this schedule, with one subject having low scores at the 50 fswg level. It is interesting to note that in those subjects with detectable VGE, the scores did not generally increase as one neared the surface. In fact, some subject's VGE scores decreased from 10 fswg to the surface.

DISCUSSION

Meaningful comparison of these schedules is difficult due to the relatively small numbers of subjects and the well established variable nature of DCS. In fact, some investigators believe that decompression outcome can be well described by the binomial probability function (14). Therefore, suitable studies are difficult to perform. Nevertheless, this report constitutes the largest series of air saturation decompressions in the literature to date. Due to the increased use of air and other nitrogen-oxygen mixtures for saturation exposures and treatments, and the potential disabling nature of DCS, it is important to attempt differentiation between the schedules presented. This will allow concentration on potentially safer schedules in future research, rather than merely confirming that seemingly unsafe schedules are indeed, unsafe.

Therefore, it is instructive to examine each schedule in light of the theoretical tissue supersaturation acheived. In Table 4, the schedules are expressed as the mean delta-P[3] (supersaturation in atmospheres - ATM) in a single half time compartment (arbitrarily chosen as the 480 minute compartment) for the entire ascent[4]. Also, the delta-P in the same half time compartment is shown for the final 5-10 fswg of ascent[5] (surfacing delta-P). Also shown in Table 4 is the decompression sickness incidence, and overall mean ascent rate for each of the four schedules.

Of the indices listed in Table 4, only the surfacing delta-P appears to correlate reasonably well with the incidence of decompression sickness symptoms. The high incidence of DCS in schedule #3 may be explained by this comparison. While the mean delta-P for this schedule is similar to the others, the surfacing delta-P is more than twice as large as the largest of the other three. Furthermore, this value is twice that allowed for this tissue by accepted (although not well validated) computations of maximal tissue nitrogen tensions (M-values) (15). Surfacing delta-Ps for the other three schedules are within these limits. It also merits mention that the same schedule as #3 was used for a series of 60 fswg air saturation dives at another Naval laboratory, and likewise gave an unacceptably high incidence of DCS (personal communication, CDR E. Thalmann, NEDU, Panama City, FL). This effect of shallow ascent rates was also shown in a recently published report (16), where 5 of 6 subects had DCS symptoms after a 60 meter nitrogen-oxygen exposure. The mean delta-P was 0.41 ATM and the surfacing delta-P was 0.37 ATM. This point is by no means clear, however, as another recent nitogen-oxygen exposure to 165 fswg (17) had a 40% bends incidence (n=10) deep to 20 fswg, where the mean delta-P was 0.26 ATM. The surfacing delta-P was 0.33 ATM and another subject developed symptoms after surfacing. Also, the SCORE exposures (6), had a very low bends incidence (<5%) when the mean delta-P was 0.88 ATM and the surfacing delta-P, 0.43 ATM. The mean ascent rates and delta-Ps for any of the above exposures do not appear

to correlate with the DCS incidence. Thus, until more data is available, reliable conclusions cannot be made. Nevertheless, the data presented here suggests that a significant slowing of the ascent rate should occur in the shallower portions (less than 20 fswg) of nitrogen-oxygen saturation decompression schedules. A reasonable goal appears to be a delta-P of about 0.5 - 0.75 ATM (480 minute compartment) for most of the decompression, gradually decreasing to about 0.20 ATM in the final 20 fsw. These conditions appear to be met by schedules #2 and #4, which gave the lowest incidence of DCS, but were also the longest.

Equation (1), from which schedules #2 and #4 were developed, is based upon a retrospective study of many saturation dives. An initial analysis of 579 helium oxygen man-decompressions indicated that the average safe rate of ascent increases linearly with the oxygen partial pressure (18). An extension of this analysis to 1179 helium-oxygen man-decompressions suggested that the average ascent rate also decreases as the saturation depth increases. Analysis of 160 man-decompressions from air or nitrogen-oxygen saturation dives indicated similar effects, but with slower ascent rates than those for helium. A recent nitrogen-oxygen exposure has also suggested that ascent rate must decrease as the saturation depth increases (17). Thus, the value of the constant k in equation (1) appears to depend upon both the depth of the saturation exposure and the inspired inert gas species.

Equation (1) appears to be a convenient empirical tool for developing saturation decompression schedules because it has only a single unknown. The schedule can be easily calculated on station, and if decompression sickness occurs, it can be made more conservative by reducing the magnitude of k.

Another factor to consider in the evaluation of these results is pulmonary oxygen toxicity. Data exists which suggests that pulmonary oxygen toxicity increases susceptibility to decompression sickness (13), although the mechanism remains unclear. It is possible that the presence of symptoms of pulmonary oxygen toxicity may have been partly responsible for the occurrence of DCS in these exposures. The degree of pulmonary oxygen toxicity in these exposures can be estimated from the forced vital capacity (FVC) measurement (19,20). Thus, one might expect that those subjects with DCS symptoms would have had greater decrements in the FVC than those without DCS symptoms. However, the FVC decrement of the five subjects with decompression symptoms in the AIRSAT exposures (the SUREX exposures represented a much lesser oxygen exposure) was not significantly different than those without symptoms. Nevertheless, it remains possible that pulmonary oxygen toxicity reduces decompression tolerance; the FVC may not be the pertinent index in this case.

Not only may pulmonary oxygen toxicity compromise decompression tolerance, it also may complicate treatment. Since current

treatment regimens for decompression sickness call for the use of hyperbaric oxygen therapy, a pre-existing degree of pulmonary oxygen toxicity may compromise tolerance of the treatment itself. This was believed to account for an unusual case of pulmonary oxygen toxicity in at least one report (2), and was the basis for modifying the treatment tables used for the cases of decompression sickness described here. It appears, however, that subjects treated with hyperbaric oxygen for decompression sickness suffer no further decrement in pulmonary function. The 6 treated cases of decompression sickness in this report had no significant change in the FVC from before to immediately after the treatment (4.85 $\pm$ .57 L versus 4.99 $\pm$ .40 L). Thus, it appears that once recovery from the toxicity begins, the effect of an oxygen treatment table is minimal.

A further factor which complicates analysis of the results in schedule #3 is the timing of the rest periods. In the AIRSAT 3 experiments, the decompression was timed so that the subjects surfaced at 1345, or just prior to the 1400 rest stop. Therefore, the subjects were traveling at the 3 feet/hr rate since 0600, from about 22 fswg all the way to the surface. Should things have been timed so that the 2400-0600 rest stop occurred between 10 fswg and the surface, the results of this schedule may have been very different. It seems reasonable that rest stops should be in relationship to depth rather than the time of the day.

The character and timing of decompression symptoms seen in this

series is similar to experiences with shallow helium-oxygen exposures (21,22). Most symptoms began while still under pressure, and the knee appears to be the most common site, indeed the only site in this series. In one reported helium-oxygen decompression series, knee pain accounted for approximately 80% of all symptoms resulting from the saturation decompression. Other symptoms commonly associated with deeper helium-oxygen saturation exposures (23), such as vestibular symptoms, were not observed in this series.

Sufficient precordial doppler monitoring for VGE was not performed to allow correlation with symptoms. However, the only subjects with type I DCS from schedules #2 and #4 also had the highest VGE score for those schedules. Overall, quantities of VGE were very low, and rarely present deeper than 20 fswg in any of the schedules. The deeper exposures had a tendency to produce detectable VGE earlier, as would be expected.

In conclusion, seven cases of decompression sickness out of 74 decompressions from nitrogen-oxygen saturation exposures at depths of 60 to 132 fswg are described in detail. Aside from the overall DCS incidence rate of 9.5%, meaningful conclusions are impossible to make in light of the statistically insufficient number of subjects. However, the data appear to suggest that the shallow ascent rates are more important in determining DCS incidence than those in the deeper portions of the decompression. Furthermore, decompression sickness in these nitrogen-oxygen

saturation exposures is similar in character and timing with that of helium-oxygen exposures. Many more decompressions, using uniform, uncomplicated criteria and procedures, will be necessary before sound concepts of decompression can be formulated.

ACKNOWLEDGEMENTS

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FIGURE LEGENDS

1. Pressurization profile for all the exposures described in this report. A: AIRSAT 1 (schedule #1), B: AIRSAT 2 (schedule #2), C: SUREX 1-6 (schedule #2), D: SUREX 7,8 (schedule #2), E: AIRSAT 3 (schedule #3), F: AIRSAT 4 (schedule #4). Clear areas represent air as the breathing media, and shaded areas represent other nitrogen oxygen mixtures; lines - 0.30 ATA oxygen, balance nitrogen; dots - 0.50 ATA oxygen, balance nitrogen.

2. Oxygen partial pressure profiles for the exposures shown in the previous figure. A: AIRSAT 1&2 (AIRSAT 2 in broken lines), B: AIRSAT 3, C: AIRSAT 4, D: SUREX 1-8 (SUREX 7&8 in broken lines).

FOOTNOTES

1. This schedule is the same one used for the Shallow Habitat Air
Dive experiment (9), with the arbitrary 8 hour sleep hold at 12
fswg deleted.

2. Modified by shortening the length of the oxygen exposure
because of encumbent symptoms of pulmonary oxygen toxicity. For
instance: the subject treated with the modified TT5 had an entire
twenty minute oxygen breathing period eliminated from both the 60
and 30 fswg stages. The TT6 had shortened oxygen breathing
periods, and lengthened
air periods.

3. This calculation is based on the equation:

$$Delta\text{-}P = ascent\ rate/((0.693/T_{1/2})(60)) - PiO_2$$

where PiO_2 is the inspired oxygen partial pressure in ATA,
delta-P is the tissue supersaturation in ATM, $T_{1/2}$ is the tissue
compartment equilibration half time in minutes (480 in this
report), and the ascent rate is in ATM/hour (ft/hr obtained by
multipying by 33).

4. The mean delta-P for the entire ascent is calculated using the

mean ascent rates from Table 4 and a time weighted average PiO_2 for the entire ascent.

5. The surfacing delta-P is based on the ascent rates for the final 10 fsw, and a PiO2 of 0.21 ATA (surface value).

TABLE 1

SUBJECT VITAL STATISTICS[*]

EXPERIMENT	N	AGE (yr)	HEIGHT (cm)	WEIGHT (Kg)	BODY FAT (%)
AIRSAT 1	11	25.5±5.0	178±4.0	77.6±7.0	19.8±4.5
(schedule #1)		(20-35)	(172-185)	(69.9-91.2)	(14.9-28.0)
AIRSAT 2	12	23.8±3.3	180±8.0	77.3±9.0	16.4±3.6
(schedule #1)		(21-32)	(162-189)	(61.7-95.7)	(11.0-22.0)
SUREX	24	26±5.0	175±6.5	75.8±8.6	13.3±4.4
(schedule #2)		(20-38)	(164-191)	(56.5-94.8)	(6.5-24.6)
AIRSAT 3	12	30.1±3.6	176±9.0	81.4±10.0	22.1±4.3
(schedule #3)		(25-36)	(162-194)	(68.4-104.4)	(15.0-27.0)
AIRSAT 4	15	28.5±6.8	175±7.8	79.6±10.7	14.3±5.4
(schedule #4)		(19-39)	(160-186)	(62-107)	(7.6-28.7)

* - units expressed as mean ± standard deviation, (range).

TABLE #2

SCHEDULES

1		2		3		4	
DEPTHS*	RATE**	DEPTHS	RATE	DEPTHS	RATE	DEPTHS	RA
60-45	10	75-70	15	132-100	12	132-50	2
45-20	15	70-60	17	100- 50	15	50-40	2
20- 5	33	60-50	19	50- 0	20	40-30	3
5- 0	36	50-40	22			30-20	3
		40-30	25			20-10	4
		30-20	30			10- 0	5
		20-10	37				
		10- 0	48				

| TOTAL | | 32:06**** | | | | | |
| TIME | 20:00 | 34:46 | | 51:42 | | 65:08 | |

Notes:

*   In feet sea water gauge (fswg).

**   In minutes per fsw.

*** Constant PiO2 of 0.50 ATA for this interval.

**** Total time for schedule from 65 fswg.

TABLE #3

VENOUS GAS EMBOLI ON ASCENT

	Depth (fswg)					
	50	30	20	10	5	0
SCHEDULE #2						
No. subjects with VGE	0	0	0	1	5	5
Mean VGE score	0/0	0/0	0/0	0/1	.4/1.9	.4/2
SCHEDULE #4						
No. subjects with VGE	1	2	3	3	–	3*
Mean VGE score	0/1	0.5/1	0.7/2	0.3/1.4	–	.7/2.1

* The one subject with DCS (see text) was treated before surfacing, and
had no detectable VGE after the treatment.

TABLE #4

SCHEDULE CHARACTERISTICS AND RESULTS

SCHEDULE	MEAN ASCENT RATE FSW/HR	DELTA-P*(T1/2=480) MEAN	SURFACING	NO. SUBJECTS	DCS SYMP
1(AIRSAT 1&2)	3.00	0.70	0.38	23	2 (8.
2(SUREX 1-8)	2.02	0.34	0.23	24	1 (4.
3(AIRSAT 3)	2.56	0.32	0.84	12	3 (25
4(AIRSAT 4)	2.03	0.29	0.16	15	1 (6.

* – Units are atmospheres (ATM)

Figure 1

ELAPSED TIME (DAY)

DEPTH FSWG

Figure 2

FILM

3-8